# Focal cortical dysplasia magnetic resonance imaging

- Stereoelectroencephalography-guided radiofrequency thermocoagulation for drug-resistant epilepsy: A meta-analysis
- Imaging Epilepsy: Past, Passing, and to Come
- Malformations of cortical development: Embryology and epilepsy
- Clinical Findings in Temporal Lobe Epilepsy Associated With Isolated Amygdala Enlargement
- Enhanced Detection of Epileptogenic Zone Using Prisma 3T MRI in Patients with MRI-negative Focal Epilepsy
- Automated Whole-Brain Focal Cortical Dysplasia Detection Using MR Fingerprinting With Deep Learning
- Converting "nonlesional" imaging occult epilepsy into a focal lesional entity using advanced imaging techniques: illustrative case
- Automated Detection and Localization of Focal Cortical Dysplasia Type II: The Optimized Detection Framework (ODF)

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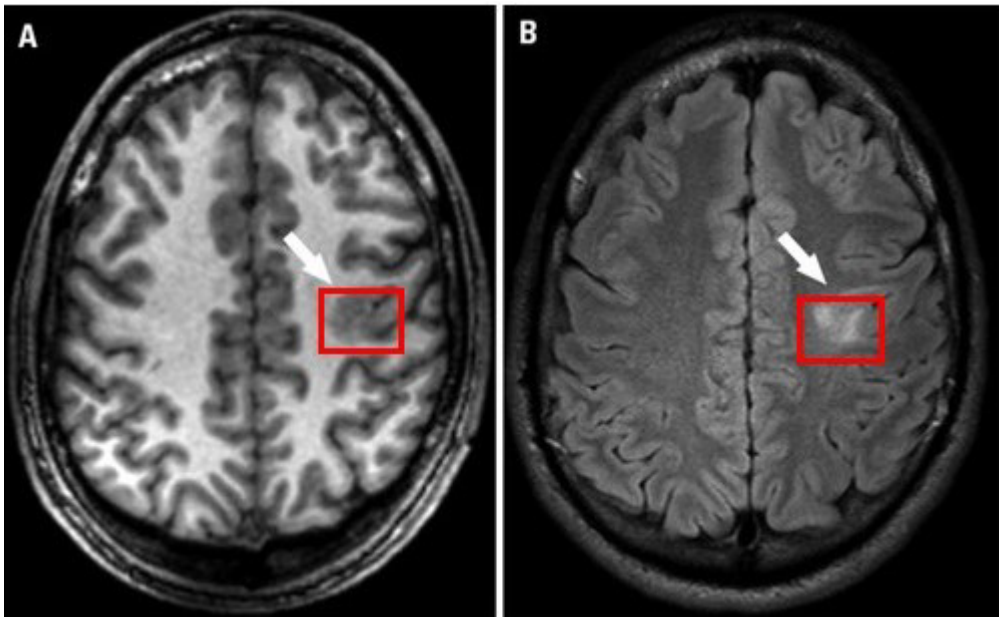
## Findings




Focal cortical dysplasia lesion (A) T1-weighted MRI and (B) FLAIR MRI

### General Features

- High-resolution MRI (preferably 3T) is essential.
- Focal cortical dysplasia Type II lesions are more conspicuous than Type I.

### Common MRI Characteristics

| Feature | Description |
|---|---|
| **Cortical thickening** | Focal or regional thickening with altered gyral pattern. |
| **Blurring of gray-white junction** | Key hallmark, especially in FCD Type IIb. |
| **T2/FLAIR hyperintensity** | Subcortical white matter hyperintensity, more evident in FCD II. |
| **Transmantle sign** | Funnel-shaped signal abnormality from cortex to ventricle (FCD IIb). |
| **Abnormal sulcation** | Shallow, widened, or missing sulci. |
| **Asymmetry** | Subtle differences compared to contralateral hemisphere. |
| **Gray matter heterotopia** | May coexist with FCD, especially Type I. |

## MRI Protocol Recommendations

- 3 Tesla scanner
- 1 mm isotropic resolution
- Volumetric T1-weighted (axial/coronal)
- T2-weighted and FLAIR in multiple planes
- Optional: DIR, PSIR, SWI

## Classification and Imaging Correlation

| FCD Type | MRI Visibility | Histological Features |
|---|---|---|
| Type I | Often subtle | Abnormal cortical layering |
| Type IIa | Easily seen | Dyslamination with dysmorphic neurons |
| Type IIb | Characteristic | Dysmorphic neurons + balloon cells + transmantle sign |
| Type III | Variable | Associated with other lesions (tumor, scar, etc) |

## Magnetic resonance fingerprinting

Ding et al. included patients with pharmacoresistant focal epilepsy and pathologically/radiologically diagnosed Focal Cortical Dysplasia, as well as age-matched and sex-matched healthy controls (HCs). All participants underwent 3D whole-brain MRF and clinical MRI scans. T1, T2, gray matter (GM), and white matter (WM) tissue fraction maps were reconstructed from a dictionary-matching algorithm based on the MRF acquisition. A 3D ROI was manually created for each lesion. All MRF maps and lesion labels were registered to the Montreal Neurological Institute space. Mean and SD T1 and T2 maps were calculated voxel-wise across the HC data. Each patient's T1 and T2 z-score maps were generated by subtracting the mean HC map and dividing by the SD HC map. MRF-based morphometric maps were produced in the same manner as in the morphometric analysis program (MAP), based on MRF GM and WM maps. A no-new U-Net model was trained using various input combinations, with performance evaluated through leave-one-patient-out cross-validation. They compared model performance using various input combinations from clinical MRI and MRF to assess the impact of different input types on model effectiveness.

They included 40 patients with FCD (mean age 28.1 years, 47.5% female; 11 with FCD IIa, 14 with IIb, 12 with mMCD, 3 with MOGHE) and 67 HCs. The DL model with optimal performance used all MRF-

based inputs, including MRF-synthesized T1w, T1z, and T2z maps; tissue fraction maps; and morphometric maps. The patient-level sensitivity was 80% with an average of 1.7 false positives (FPs) per patient. Sensitivity was consistent across subtypes, lobar locations, and lesional/nonlesional clinical MRI. Models using clinical images showed lower sensitivity and higher FPs. The MRF-DL model also outperformed the established MAP18 pipeline in sensitivity, FPs, and lesion label overlap.

The MRF-DL framework demonstrated efficacy for whole-brain FCD detection. Multiparametric MRF features from a single scan offer promising inputs for developing a deep-learning tool capable of detecting subtle epileptic lesions [1)].

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Ding et al. aim to improve detection of Focal Cortical Dysplasia (FCD) in patients with pharmacoresistant focal epilepsy using Magnetic Resonance Fingerprinting (MRF) combined with deep learning (DL). The motivation is compelling, given the clinical challenge of identifying subtle or MRI-negative FCD lesions and the limitations of existing morphometric tools like MAP18.

 Strength: Tackles a well-known diagnostic gap using an innovative approach (MRF-DL).

⚠ Caution: While promising, MRF is not yet a standard clinical tool, potentially limiting immediate generalizability.

2. Study Design and Methods

Participants:

40 patients with histologically or radiologically confirmed FCD

67 age- and sex-matched healthy controls (HCs)

Imaging Protocol:

3D whole-brain MRF and standard clinical MRI

Extraction of quantitative T1, T2, GM, and WM fraction maps via dictionary-matching

Voxel-wise Z-scoring relative to the HC database

Manual ROI lesion segmentation and registration to MNI space

U-Net trained using combinations of MRF-derived maps

 Strengths:

Inclusion of multiple FCD subtypes (IIa, IIb, mMCD, MOGHE) enhances generalizability.

Use of z-score normalization relative to a well-matched control set is statistically sound.

Lesion masking and MNI registration support spatial consistency.

⚠ Limitations:

Manual ROI definition may introduce inter-rater variability, which isn’t addressed.

No external validation cohort or site-to-site reproducibility analysis.

Leave-one-patient-out cross-validation may overestimate generalizability in larger clinical datasets.

3. Results and Performance Sensitivity: 80% for the best-performing MRF-DL model

False Positives: 1.7 per patient on average

Performance was superior to both clinical MRI models and the MAP18 pipeline, even in MRI-negative cases.

 Strength: Demonstrates that MRF-based models capture subtle morphometric and relaxometric abnormalities beyond conventional imaging.

⚠ Caution:

The 1.7 FP rate per patient may still be problematic for clinical workflows (e.g., surgical planning).

No discussion on model explainability or heatmaps—important for clinical trust.

4. Interpretation and Innovation This is one of the first studies to leverage quantitative MRF-derived inputs for DL-based FCD detection, achieving notable performance improvements over standard tools.

 Innovations:

Synthesized structural images (MRF-T1w) from MRF data

Joint use of tissue maps and morphometric z-scores

Single acquisition protocol with multiparametric output

⚠ Reservations:

Requires access to MRF, which is not widely available.

Training data size is relatively small for DL (~40 patients), potentially limiting the network’s robustness to variability in lesion appearance.

5. Conclusions and Clinical Implications The MRF-DL framework provides a promising direction for non-invasive, quantitative, and automated FCD detection, especially in challenging MRI-negative cases.

 Implication: Could reduce time to diagnosis and improve presurgical planning.

 Next Steps:

Validation across centers and MRI platforms

Testing in larger, real-world datasets

Integration into clinical decision-support systems

Final Assessment Ding et al. present a technically sound and innovative study with high clinical

relevance. While limitations exist regarding sample size, generalizability, and clinical implementation hurdles, the findings significantly advance the potential of MRF-based deep learning in the automated detection of epileptogenic lesions.

Overall Rating: ★★★★☆ (4/5)

 Innovative and promising, but early-stage with key limitations in generalizability and clinical readiness.

## Diagnostic Tips

- Use advanced post-processing (e.g. voxel-based morphometry).
- Co-register with PET or SPECT to enhance detection.
- Multidisciplinary review increases diagnostic yield.

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In type I cortical dysplasia, MR imaging is often normal. Also, in both types, the lesion seen on MRI may be smaller than the seizure-generating region seen in the EEG. The abnormalities may also involve vital for life brain parts, where curative surgery will not be an option. Therefore, other diagnostic imaging techniques such as FDG PET, MEG, DTI, and intra-cranial EEG are widely used to establish the diagnosis and to decide on management [2].

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In many cases of FCD, and particularly in Type II, the MRI is abnormal, showing an abnormally bright focal area on T2 and FLAIR sequences, which often has a characteristic “tail” extending to the margins of the ventricles. However, the MRI can be normal in some cases, particularly with Type I FCD. In these cases, the diagnosis can only be made after the removal of the brain region that causes the seizures and detailed examination under the microscope.

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High-resolution MRI. Extremely good for detecting neuronal developmental abnormalities (e.g. cortical dysplasia) that may produce complex partial seizures (CPS) [3].

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Morphometrics and PET/MRI co-registration show promising results in identifying subtle focal cortical dysplasia (FCD) abnormalities in cingulate epilepsy (CE) with negative results on conventional MRI, which can be otherwise challenging. More importantly, a combination of MRI post-processing and PET/MRI co-registration can greatly improve the identification of epileptic abnormalities, which can be used as a surgical target. MAP and PET/MRI co-registration should be incorporated into the routine presurgical evaluation [4].

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Electroencephalography (EEG)-functional magnetic resonance imaging (fMRI) and magnetoencephalography are helpful in guiding electrode implantation and surgical treatment, and high-frequency oscillations help to define the extent of the epileptogenic dysplasia.

Automated surface-based MRI morphometry equipped with machine learning showed robust performance across cohorts from different centers and scanners. The proposed method may be a valuable tool to improve FCD detection in presurgical evaluation for patients with pharmacoresistant epilepsy [5].

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Ultra high-field MRI has a role in understanding the laminar organization of the cortex, and 18F positron emission tomography (FDG-PET) is highly sensitive for detecting FCD in MRI-negative cases. Multimodal imaging is clinically valuable, either by improving the rate of postoperative seizure freedom or by reducing postoperative deficits. However, there is no level 1 evidence that it improves outcomes. Proof for a specific effect of antiepileptic drugs (AEDs) in FCD is lacking. Pathogenic mutations recently described in mammalian target of rapamycin (mTOR) genes in FCD have yielded important insights into novel treatment options with mTOR inhibitors, which might represent an example of personalized treatment of epilepsy based on the known mechanisms of disease. The ketogenic diet (KD) has been demonstrated to be particularly effective in children with epilepsy caused by structural abnormalities, especially FCD. It attenuates epigenetic chromatin modifications, a master regulator for gene expression and functional adaptation of the cell, thereby modifying disease progression. This could imply lasting benefit of dietary manipulation. Neurostimulation techniques have produced variable clinical outcomes in FCD. In widespread dysplasias, vagus nerve stimulation (VNS) has achieved responder rates >50%; however, the efficacy of noninvasive cranial nerve stimulation modalities such as transcutaneous VNS (tVNS) and noninvasive (nVNS) requires further study. Although review of current strategies underscores the serious shortcomings of treatment-resistant cases, initial evidence from novel approaches suggests that future success is possible [6].

FDG-PET should be registered on three-dimensional MRI for better detection of focal cortical dysplasia [7].

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A study shows a high level of concordance between FCD and BOLD response. This data could provide useful information especially for MRI negative patients. Moreover, it shows in almost all FCD patients, a metabolic involvement of remote cortical or subcortical structures, corroborating the concept of epileptic network [8].

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